Modern Concepts of Cardiovascular Disease

Published monthly by the AMERICAN HEART ASSOCIATION

1790 BROADWAY AT 58TH STREET, NEW YORK, N. Y.

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UNIVERSITY OF COLORADO SCHOOL OF MEDICINE

VOL. XVI NOVEMBER, 1947 No. 11

THE BACKWARD FAILURE THEORY OF CONGESTIVE HEART DISEASE

Definition

The chief manifestations of congestive heart failure are cardiac enlargement, diminished vital capacity with dyspnea (due to failure of the left side of the heart) and increased venous pressure with visceral congestion (due to failure of the right side of the heart). The "backward failure" theory is that concept of congestive heart failure which ascribes these major clinical phenomena to an accumulation of blood in those vascular areas which drain toward the failing chamber or chambers of the heart. This conception of the cause of congestive heart failure was first suggested by Raymond Vieussens in 1715 and was clearly formulated by James Hope in 1842.

Physiology of the Failing Heart

The classical studies of Starling and his coworkers developed the basis for our modern conception of the cardiodynamics of the failing heart. The "law of the heart," as postulated by Starling, states that the mechanical energy set free during cardiac contraction depends on the length of the muscle fibers.

When disease processes of the myocardium are present, incomplete emptying of the cardiac chambers occurs, and the increase in residual blood leads to dilatation of the heart. A diseased heart can maintain a given cardiac output, but only provided the length of the fibers is increased; therefore, a diseased heart must dilate in order to maintain a constant minute volume. Although dilatation of moderate degree may allow a weakened myocardium to maintain an output near the normal level, dilatation of extreme degree, such as may occur in the most advanced stage of congestive failure, may stretch the muscle fibers beyond their optimal length and cause the output to decline markedly. Under such circumstances the manifestations of forward failure (feebleness, decline in blood pressure and so forth) are superimposed on those of backward failure.

As the amount of residual blood increases in the ventricle, dilatation occurs, and with this the entrance of blood in diastole eventually is impaired. The auricle then tends to accumulate residual blood until such a time as its own mean pressure rises to a sufficient extent to restore an effective pressure filling gradient between auricle and ventricle. Since the size of both cavities is now greater, a given inflow may be maintained with a lower gradient of pressure. Thus the dynamics of ventricular filling may be partially or (less commonly) entirely restored, but only at the expense of a lasting increase in the mean intra-auricular pressure.

Normally the gradient between auricular pressure and peripheral venous pressure is such that a lower pressure exists in the former which allows auricular filling with relative ease. With the elevation of auricular pressure consequent to failure of the heart, however, this gradient is lowered, so that auricular filling is impaired. This in turn produces stasis in the great veins and leads to an increase in systemic venous pressure. It must be recognized that although the velocity of blood flow is diminished in patients with heart failure, this does not imply that the volume of flow (that is, the cardiac output) is necessarily correspondingly decreased. Careful measurements have shown repeatedly that the volume of blood in the pulmonary and systemic vascular systems is increased in congestive heart disease. Thus, although the speed of circulation of blood is markedly decreased, the increased size of the stream bed may permit the total blood flow per unit of time to be maintained at levels not far below normal. These events in turn lead to an engorgement of the capillaries and an increase in intra-capillary pressure. The increase in the venous and capillary pressures in turn gives rise to most of the clinical phenomena of congestive failure. Even though the average veno-auricular pressure filling gradient is markedly diminished in failure of the right side of the heart, a corresponding decline in the flow of blood from the peripheral veins to auricle need not occur, because the greater volume of the stream bed permits a given flow to occur at a lower pressure gradient.

ANNUAL MEETING

The Annual Meeting and Twenty-first Scientific Sessions of the American Heart Association will be held in Chicago, Illinois, on June 18th and 19th, 1948. The Hotel Stevens will be the headquarters for all meetings. The first Scientific Session will be held on Friday afternoon, August 18th. The Annual Dinner is scheduled for Saturday evening, June 19th, at the Stevens. Hotel rooms will be in great demand and every member who wishes to attend is urged to make reservations immediately. Please see the following page for instructions regarding reservations received from the Reservations Manager of the Stevens and addressed to Associations meeting prior to the American Medical Association.

The backward failure concept holds, therefore, that a disturbance of the veno-auriculo-ventricular pressure filling gradients is the fundamental disturbance which is causative of the clinical symptoms and signs of classical congestive heart failure. The significance of the increased peripheral venous pressure in disease of the heart recently has been clarified further both by work on animals and on human beings. It recently has been shown that when the acutely injured heart of the dog is unable to cope with an increased burden, it responds by an increase in venous pressure. Measurements of auricular and ventricular pressures (by the method of intracardiac catheterization), as well as of peripheral venous pressures, in patients with congestive heart failure have recently been reported. Whenever a significant degree of failure was present, diastolic pressure was elevated in the right ventricle, and the mean auricular pressures were found to be increased. Furthermore, peripheral venous pressures were elevated in most cases, and a reduction of the normal veno-auricular pressure filling gradient was a prominent finding in the patients with heart failure. Indeed the peripheral venous pressure, mean right auricular and mean right ventricular diastolic pressures frequently corresponded closely, and all were usually elevated. In failure of the left side of the heart, the right ventricular systolic and pulse pressures were elevated in all cases. These direct measurements of the prevailing intracardiac and venous pressures in patients with failing hearts constitute direct evidence that the dynamic changes postulated by the backward failure hypothesis do in fact occur.

Results of Studies of Cardiac Output

When the results of studies of cardiac output are considered, it must be recognized that all methods in use have some possible sources of error. Studies made by the older methods (acetylene, ballistocardiograph and so forth) led to the following conclusions:

1. The minute output of the heart is usually subnormal in patients with heart disease and diminished cardiac reserve. 2. Normal persons usually have normal cardiac outputs, but an occasional person without symptoms or signs of heart disease has an output as low as that of most of the patients with cardiac disturbance. 3. In general, patients with early congestive failure have cardiac outputs within the same range as those of patients with cardiac disease but without congestive phenomena. 4. An occasional patient with congestive heart failure has a cardiac output per minute greater than normal, and in exceptional instances the values for cardiac output in proportion to consumption of oxygen may be elevated. 5. Clinical improvement may be found to be associated with an increase, a decrease or no change in the cardiac output. Numerous investigators have reported results in accord with these conclusions. A few investigators, on the contrary, have found a close correlation between the diminution of cardiac output and the severity of the congestive manifestations, and some have reported that clinical improvement was regularly associated with a rise in cardiac output per minute.

Reports recently have been made on patients with heart disease who have been studied by the direct Fick method after intracardiac catheterization. Richards and his co-workers recently reported data obtained by this method, from patients with rheumatic, arteriosclerotic and pulmonary heart disease in failure. In the patients with rheumatic heart disease, the reduction in cardiac output was more marked than by the older methods of measurement; the difference possibly was due to the fact that most of the studies with the catheter technic have been done with the patient recumbent, whereas the studies with foreign-gas methods were made with the patient in the sitting position. In one patient with arteriosclerotic heart disease, the cardiac output was moderately diminished (cardiac index 2.33) when a relatively good state of compensation was present. Further studies on this patient later, during advanced decompensation, however, showed a diminution of cardiac index only from 2.33 to 2.01. Stoke volumes were markedly decreased in these patients. By contrast, patients with pulmonary disease and definite evidences of isolated failure of the right side of the heart were studied by the same method employed on the patients with rheumatic and arteriosclerotic heart disease, and were found to have cardiac outputs which were essentially within normal limits (average cardiac index for this latter group 3.45). Somewhat similar findings have been reported by others, groups of patients with congestive failure presenting low and occasionally high cardiac outputs respectively.

Under certain circumstances patients with congestive failure may present the clinical signs of increased cardiac output, including moderate tachycardia, bounding pulse, elevated pulse pressure, palpitation and exaggeration of the intensity of the heart sounds. Such findings are encountered in persons with congestive failure due to thyrotoxicosis, severe anemia or beriberi and may occur acutely when persons with previously compensated cardiac disease of any type are given massive intravenous infusions. Cases of this type are rare but are of considerable theoretical importance because they indicate that a diminution in the output of the heart is not a necessary prerequisite for the development of the manifestations of congestive heart failure. The latter, therefore, can be logically ascribed only to increases in venous pressure (and its attendant sequelae) in the pulmonary and systemic circulations.

These results are readily understood if it is remembered that the cardiac output depends primarily on (1) the venous (filling) pressure, and (2) the myocardial capacity. Thus in the early, asymptomatic stage of heart disease, there is relatively little change in cardiac output. In patients whose tolerance for exercise is diminished but who are free of congestive failure, the venous pressure is normal but myocardial function is impaired; hence the stroke volume and cardiac output tend to decrease. With further weakness of the myocardium, the venous pressure rises and the symptoms of venous congestion appear; further marked decline in cardiac output (in the stage of moderately advanced failure) is prevented, however, by the compensating effect of the elevated venous pressure, which acts to increase the cardiac output. Thus it will be seen that the development of early congestive failure is usually not attended by a striking decrease in cardiac output. In extreme and advanced cardiac failure, as in agonal states, it appears probable that a further diminution of cardiac output occurs, although few dependable data are available on this point.

Relationship of Physiologic Alterations to Symptoms

When the picture of acute failure of the heart is considered, it must be recognized at once that this is an entirely different clinical picture from that of classical "congestive failure" as defined previously. True forward failure of cardiac origin is that abnormality of the circulation in which, as the result of a primary disturbance in the heart, there is an inadequate blood supply to the tissues, which produces a symptom-complex resembling that of shock (peripheral circulatory failure) and characterized especially by the sudden onset of feebleness of body and mind, sometimes progressing to unconsciousness, coldness of the skin and decline in blood pressure. This syndrome may be caused by (1) sudden weakness of the myocardium (as in myocardial infarction or diphtheritic myocarditis); (2) by neurogenic influences (as in the asystole of carotid sinus hypersensitivity); (3) by mechanical defects (as in ball-valve thrombus) or (4) by severe tachycardia,

with diminished cardiac filling and impairment of myocardial nutrition due to diminished coronary flow. The underlying circulatory defect in the causation of the clinical manifestations of all these states is a severe and abrupt diminution in blood flow to the tissues of the body.

The clinical picture of congestive failure, however, which is the subject of this discussion, is an entirely different one from that of true forward failure. Failure of the left ventricle with resultant elevation of left auricular pressure causes stasis of blood in the lungs, which in turn gives rise to increased rigidity of the pulmonary tissues. Vital capacity declines, and measurements of the intrapleural work performed reveal that a disproportionately large amount of energy is expended to achieve adequate ventilation. It also has been shown that the stimulation of breathing in patients with cardiac failure is in the main, of reflex, rather than chemical origin, and that of the several reflexes concerned, the most important one arises in the lungs and is caused by the effect of congestion on the afferent vagal endings. These same alterations in respiration and their recognition at cortical sensory levels together constitute the fundamental physiologic alterations seen in the clinical state of dyspnea.

The concept that pulmonary edema is caused by forward failure with diminished blood flow to the lungs (suggested by James MacKenzie) has been proved to be untenable. Progressive constriction of the pulmonary artery fails to precipitate respiratory abnormalities which resemble dyspnea. The occurrence of isolated left ventricular failure, with severe dyspnea without peripheral edema, is one of the most convincing proofs of the backward nature of congestive heart failure.

When the right heart fails, pressure rises in the systemic veins, and visceral engorgement and edema tend to result. The development of edema in congestive heart failure is apparently due, primarily, to an increase in hydrostatic pressure in the venocapillary loop, but the extent of the fluid accumulation which occurs is dependent on other factors as discussed later. That this increased venocapillary pressure is associated with an increased blood volume in chronic heart failure is generally agreed on by most investigators. It would seem that this increase in blood volume is one of the most important alterations in congestive failure but the mechanism of its production is not entirely clear. It has been proposed that the hypervolemia is due to a defect in renal excretion of sodium but this hypothesis does not account for the increase in the volume of erythrocytes. It has been shown that when edema is produced in cases of cardiac disease by increasing the intake of sodium chloride, the increase of retention of fluid by the tissues and the increase of blood volume occur first; the venous pressure rises subsequently. When, however, congestive failure is induced by withdrawing digitalis from patients with auricular fibrillation, a definite rise in venous pressure occurs before the patients gain weight, and this observation suggests that the presence of backward failure with an elevation of venous pressure is the initial factor in the production of cardiac edema. On the other hand this group of patients develop only minimal edema unless the intake of sodium is increased, and this observation supports the concept of the importance of retention of sodium as a quantitative (although apparently secondary) factor of great importance in the production of cardiac edema.

The relative importance of the tissue factor (increased venous pressure) versus the renal factor (retention of sodium) in the production of cardiac edema is illustrated by patients with obstruction of the superior vena cava. Here edema of the upper portion of the body is the rule but the degree of edema is almost invariably slight. It is clear that well-marked generalized edema cannot occur unless the kidneys retain sodium excessively and the renal factor, therefore, may be regarded as the quantitatively more important factor in cardiac edema. On the other hand the demonstration that the venous pressure rises first when digitalis is withdrawn, plus the observation that increased venous pressure per se may cause slight edema in the absence of any renal impairment, seems to indicate clearly that the primary factor (that is, the qualitative factor which distinguishes cardiac from other forms of edema) is elevation of venous pressure.

It has been shown that the renal blood flow and glomerular filtration rate are reduced in congestive failure, and the suggestion has been made that the decrease in renal blood flow is the result of decline in cardiac output. However, in view of the fact that typical congestive failure may occur in the presence of a normal or elevated cardiac output, it would appear that the decline in renal blood flow (in such cases at least) must be ascribed to local renal vasoconstriction as the result of some unknown humoral or nervous mechanism. To designate such a local disturbance as forward failure of the heart seems to the writers to require an unwarranted extension of the idea of heart failure to include all vasospastic states in which there is local decline in blood flow.

The demonstration that reabsorption of sodium proceeds at a relatively normal level in the presence of congestive failure despite the marked decline in renal blood flow has led to the hypothesis that the reduction in renal blood flow is the cause of the retention of sodium and of edema. This concept, however, fails to explain the absence of edema in many cases in which renal blood flow is markedly reduced (for example in cases of malignant nephrosclerosis), and its absence in the dog with experimental renal ischemia. Moreover recent studies using radioactive sodium suggest that the degree of decline in excretion of sodium may far exceed the degree of decline in glomerular filtration and hence that excessive reabsorption rather than diminished filtration of sodium may be the major factor.

It would seem, therefore, that in patients with congestive heart failure, the primary disturbance tending to cause edema appears to be the increase in venous pressure. However, in order for a significant degree of edema to occur it is necessary that the renal excretion of sodium be impaired, and this mechanism is the important quantitative determinant of edema; the increase in venous pressure is the primary and qualitative factor which distinguishes cardiac edema from most other types of edema. The role of the kidney in determining retention of sodium, thereby causing further elevation of venous pressure, and aggravating the tendency toward edema, cannot be doubted. It seems certain that changes in renal function are important in the causation of cardiac edema; the exact nature of these changes, however, must await further clarification.

Summary

The symptoms of classical congestive heart failure are believed to be due to the increased venous pressure (with its attendant train of consequences in the greater and lesser circulation) which develops in the venous reservoirs draining toward the cardiac chambers during cardiac failure. Slight, moderate, or severe diminution in cardiac output usually occurs with heart failure, and this functional change can account for the lassitude, fatigability and faintness which patients sometimes display. However, there seems to be no valid evidence for the view, once widely held, that dyspnea, the cardinal symptom of congestive failure, is related to diminished cardiac output. True forward failure of the heart, which is attended by severe and usually acute lowering of cardiac output, produces a characteristic symptom-complex of a shocklike nature, and this state is actually due to diminished blood flow to the tissues. Congestive heart failure, however, is an entirely

different clinical picture, which may occur in the presence of a low, normal, or elevated cardiac output, and may even occur when the output is high in relation to the oxygen consumption of the body. Since even under the latter circumstances dyspnea and edema may be present, it is clear that these symptoms cannot be primarily the result of diminished output. Instead, they must be regarded as primarily related to the decline in veno-auriculo-ventricular filling gradient, and to the elevation of venous pressure, which occur regularly in congestive heart failure, rather than as the result of the changes in cardiac output which occur inconstantly. Retention of sodium occurs in congestive heart failure (as a result of a defect in renal function as yet not wholly clarified) and this acts as an important aggravating factor in the production of edema.

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TO ASSOCIATIONS MEETING PRIOR TO THE AMERICAN MEDICAL ASSOCIATION:

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Please indicate arrival date upon which you will come into the hotel and a departure date, as well as approximate time—A.M., P.M., or Evening. These reservations will be accepted for a period ending on Sunday during the day.

Those who come in for a stay carrying over beyond Sunday, June 20, 1948, (checkout hours, Noon to 3:00 P.M.) and into Monday, Tuesday, or later, are asked to indicate when they will leave.

In all cases, when such have indicated later departure dates, their applications will be processed by the American Medical Association or their Housing Committee as may be later organized.

If they are approved as members of the American Medical Association, their departure date will be honored in the hotel.

If they are not approved, the reason being that they may not be members of the Association or if they do not indicate the departure date, the reservation will be confirmed only up to Sunday Noon or afternoon as above set forth.

We shall appreciate your complete cooperation to work out this plan successfully.

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